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Gravity Perception: How Plants Stand up for Themselves

Dispatch

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How organisms sense and respond to gravity is a long-standing question in biology. Recent studies of plant shoot gravitropism support a new physical model of gravity perception.

Gravity provides a basic polarity for the development, growth and environmental response of plants. Shoots grow up toward a source of light, while roots grow down toward sources of water, nutrients and structural support. Though plant gravitropism has been a topic of research for two centuries [1], the molecular nature of the gravity signaling pathway remains largely unknown. Recent genetic and live imaging studies of shoot gravitropism in the model plant *Arabidopsis thaliana* have provided new insights into this intriguing phenomenon.

Gravitropism can be divided into three stages: perception of the gravity vector; transduction of the signal; and bending of the responding organ in the appropriate direction. In plants, gravity perception relies upon the downward movement of amyloplasts, specialized plastids packed with heavy starch granules [2]. The subsequent bending of organs is mediated by the plant growth hormone auxin. Auxin promotes cell elongation in the shoot, but inhibits it in the root; thus, an increase in auxin levels at the bottom of a reoriented shoot results in upward bending, while accumulation in the bottom half of a root causes downward bending. Current theory holds that rapid re-localization of a plasma-membrane-localized auxin transporter to the new bottom side of responding cells transduces the gravity signal by directing lateral auxin transport to cells just below [3]. However, the molecular mechanism by which the downward movement of amyloplasts directs the re-localization of auxin tranporters remains unknown.

In *Arabidopsis* shoots, gravity perception occurs in the endodermis, a cylindrical layer of cells that extends the length of the shoot (Figure 1). Mutants that do not specify the endodermis properly, or do not develop endodermal amyloplasts, or that cannot synthesize amyloplast starch, are defective in shoot gravity response [4–6]. Furthermore, the artificial displacement of amyloplasts with high-gradient magnetic fields was sufficient to direct the bending of *Arabidopsis* shoots [7]. Though some data suggest the existence of a second, amyloplast-independent gravity-sensing pathway (for example, see [8]), it is clear that amyloplast movement is critical for a robust gravity response in *Arabidopsis* shoots.

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Genetic studies in Arabidopsis have proved useful in the study of shoot gravity signaling. The shoots of the shoot gravity response (*sgr*) mutants are unable to respond to a change in gravity vector, but are capable of a normal phototropic response [9]. In recent studies [10–13], Tasaka and colleagues have characterized the *sgr2*, *sgr3* and *sgr4/zig* mutants and the affected genes. *SGR4/ZIG* encodes a v-SNARE, AtVTI11p, while *SGR3* encodes a t-SNARE, AtVAM3p. v-SNAREs are localized to vesicle membranes, and are thought to mediate membrane trafficking and vesicle fusion through interaction with cognate t-SNAREs on the target organelle. In plant cells, AtVTI11p is localized to the *trans*-golgi network and the prevacuolar compartment, and AtVAM3p is localized to the prevacuolar compartment and the vacuole. Both proteins are found in a complex in endodermal cell extracts [11,14]. Taken together, these results implicate SGR3 (AtVAM3p) and SGR4/ZIG (AtVTI11p) in protein trafficking from the golgi to the vacuole in endodermal cells of the *Arabidopsis* shoot.

How does protein trafficking to the vacuole contribute to gravity sensation in the shoot? A hint comes from analysis of the ultrastructure of endodermal cells from wild-type and *sgr* mutant shoots [11,15]. In wild-type plants, transmission electron microscopy reveals that most of the endodermal cell volume is taken up by the vacuole, and amyloplasts are located in the bottom portion of the cell, suspended within transvacuolar strands of cytoplasm (Figure 2A). In *sgr3* and *zig* mutants, an accumulation of abnormal vacuolar or vesicular structures is observed in endodermal cells (Figure 2B). Furthermore, amyloplasts are not restricted to the bottom portion of the cell but are distributed around the cell periphery, and are no longer associated with transvacuolar strands. These mutants are likely agravitropic because they are defective in the first step in gravity sensation, the sedimentation of amyloplasts.

Tasaka and colleagues speculate that an unknown factor required for the appropriate association between

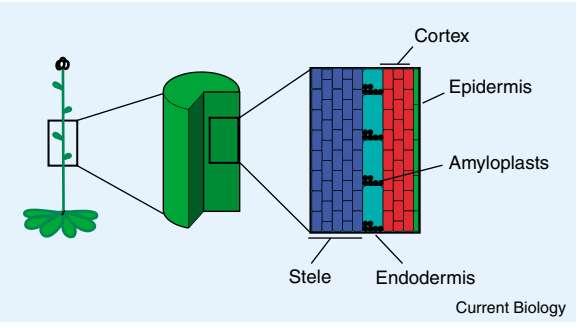

Figure 1. Structure of the *Arabidopsis* shoot.

Gravity perception in the shoot requires the endodermal cell layer. Amyloplasts, specialized plastids harboring starch granules, are located in the endodermal cells.

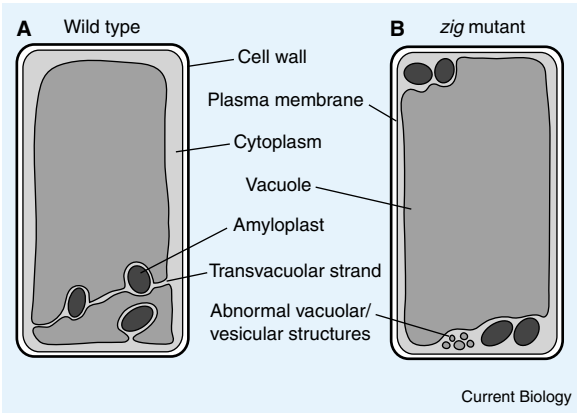

Figure 2. Ultrastructure of shoot endodermal cells.

(A) In wild-type shoot endodermal cells, amyloplasts are found in transvacuolar strands and are localized to the bottom of the cell. (B) In *sgr4/zig* mutants, amyloplasts are found at both the top and bottom of the shoot endodermal cells and are not associated with transvacuolar strands. Abnormal vacuolar or vesicular structures are also observed.

amyloplasts and the vacuole relies upon SGR3 and SGR4/ZIG for proper subcellular localization. A good candidate for such a factor is SGR2 [10], as *sgr2* mutants have similar vacuole and amyloplast abnormalities to *sgr3* and *zig* mutants, and as a SGR2–GFP fusion protein was found to localize to the vacuolar membrane. SGR2 is a member of the PA-phospholipase A1 family, and may thus be involved in changing membrane structure, fluidity or function, or in the production of second messenger molecules.

These data indicate that amyloplast sedimentation itself is a complex phenomenon. Real-time imaging of endodermal cells harboring a plastid-targeted GFP transgene revealed that amyloplasts undergo continuous, highly dynamic movement. These saltations are dramatically reduced in *sgr4/zig* mutants (M. Tasaka, personal communication). Thus, amyloplasts do not drop by default to the bottom of the cell; rather, their position appears to be controlled by association with other structural components of the cell. In fact, amyloplasts in the shoot may also interact with the cytoskeleton, as preliminary experiments show a reduction in amyloplast movement when shoots are treated with the actin polymerization inhibitor latrunculin B. Interestingly, latrunculin B treatment does not abolish, but rather dramatically enhances, gravitropic curvature of the shoot [16].

These data can perhaps be best understood in terms of the tensegrity model of cell architecture [17,18]. According to this model, a local stress can induce both local and global structural responses in the rest of the cell through adjustments in the continuous tension provided by the cytoskeleton. Though initially developed to explain the behavior of cells responding to tensions generated by the extracellular environment, this concept may also be applied to plant cells responding to tension generated from within the cell.

Amyloplast movement may be constrained by a global tensegrity network, generated by interplay between the cytoskeleton, gravity, the vacuole and perhaps other forces such as turgor pressure. When the vector of gravity changes, downward movement of the relatively dense amyloplasts produces a localized stress on the network. These localized tensions might then produce a global cellular response, perhaps activating mechanosensitive receptors in the vacuolar or plasma membrane, and culminating in the asymmetric localization of auxin transporters. This amyloplast-generated disruption of the tensegrity network might be simulated by treatment with latrunculin B, resulting in an enhanced gravitropic response.

It is not clear if the same model can be applied to gravitropism of the root. The roots of *sgr* mutants respond normally to gravity, and the gravity responsive cells of the root do not have the large vacuoles found in the shoot endodermis. Instead, the amyloplasts in the root columella cells appear to interact with patches of unusually shaped, 'nodal' endoplasmic reticulum [19]. How the same signal (amyloplast sedimentation) concludes with the same output (asymmetric auxin distribution) in the different structures of the root and the shoot remains a very intriguing question. The implementation of molecular genetic and live cell imaging approaches should improve our understanding of this process in the future.

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